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**KINETICS AND KINEMATICS OF THE KNEE DURING A SINGLE LEG SQUAT 3-10 YEARS AFTER AN INTRA-ARTICULAR KNEE INJURY SUSTAINED WHILE PARTICIPATING IN YOUTH SPORTS**

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**Purpose:** Osteoarthritis (OA) is the most common joint disease and the most common form of arthritis. The cause of OA is complex and thought to result from the interplay of a variety of risk factors including joint injury and obesity. Prospective studies report a 10-fold increased risk of post-traumatic osteoarthritis (PTOA) 12-20 years after knee injury. Within this high-risk group, it is unknown whether altered knee joint kinematics and kinetics post-injury are associated with accelerated onset and/or progression of PTOA. The single leg squat (SLS) is a common functional movement used to assess lower limb mechanics. Previous studies have identified biomechanical changes during a SLS in patients with OA, however there is a lack of information related to SLS biomechanics early in the period (< 10 years) between joint injury and disease onset. Thus, the objective of this study is to compare knee joint kinematics and kinetics during the SLS between those with a youth sport-related intra-articular knee injury sustained 3-10 years previously and healthy age, sex, and sport matched controls.

**Methods:** This historical cohort study includes thirty individuals with a sport-related intra-articular knee injury sustained 3-10 years previously (median: 6.4 yrs) and thirty uninjured age, sex, and sport matched controls (32 males, 28 females; 15-26 yrs). History of intra-articular knee injury (clinical diagnosis including bone, cartilage, ligament or meniscal injury requiring medical attention and time loss from sport) was established from previous study injury report forms and then confirmed by participants. Participants performed 15 SLS (3 trials per limb, 5 squats per trial) to approximately 45° of knee flexion. Three-dimensional marker data (3 markers per limb segment) were captured at 240 Hz with a high-speed eight-camera motion analysis system (Motion Analysis Corporation, Santa Rosa, California, USA). The RMS error in the motion analysis system has been reported as 1.5mm and 1.8 degrees. The ground reaction forces were collected with one force plate (2400 Hz; Kistler 9287, Kistler Instruments AG, Winterthur, Switzerland). Kinematics and kinetics were calculated with an in-house inverse dynamics Matlab program (Matlab 2013a, Mathworks, Natick, Massachusetts, USA). Outcome measures include bilateral knee adduction moment and knee angles in all three planes (valgus/varus, flexion/extension, and internal/external rotation). Descriptive statistics (mean within-pair difference, 95%CI) were used to compare study groups.

**Results:** Injured participants demonstrated increased knee valgus on both the index [mean within-pair difference, (95%CI); 2.63° (0.93, 4.34)] and non-index knee [2.75° (0.53, 4.97)] compared to age, sex, and sport matched controls. The mean absolute valgus angles for each of the groups are summarized in figure 1. There were no between group differences in the knee flexion-extension angle [index; -0.52° (-4.31, 3.28), non-index [-0.29° (-4.28, 3.70)], internal-external rotation angle [index; -1.13° (-3.46, 1.20), non-index [0.07° (-2.00, 2.13)], or the adduction moment of the knee [index; -15.47 Nm (-39.11, 8.18), non-index [-18.48 Nm (-58.26, 21.31)].

**Conclusions:** This study provides preliminary evidence of larger bilateral knee valgus angles during the SLS in young adults with a sport-related knee injury that occurred 3-10 years previously, compared to age, sex and sport matched controls. These results are consistent with current literature concerning biomechanical changes after a knee injury and the progression of disease in those with OA. To our knowledge this is the first time that these differences have been documented in the interval between joint injury and disease onset. A larger sample will allow for comparison of a greater number of outcome variables between study groups, including trunk and hip biomechanics, as well as their association with PTOA onset and progression.

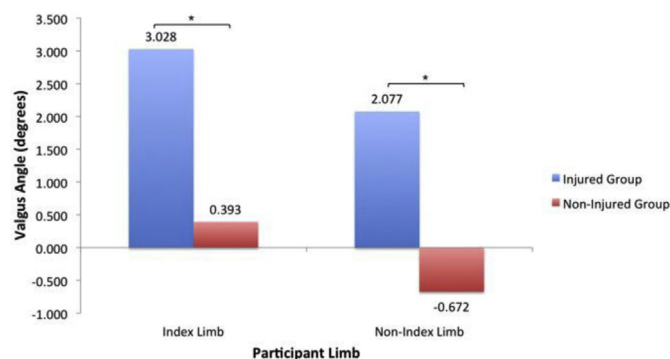


Figure 1. Valgus Angle by Injury History and Limb (\* denotes significance at a 95% confidence level).

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**NEW INSIGHT IN THE RELATIONSHIP BETWEEN REGIONAL PATTERNS OF KNEE CARTILAGE THICKNESS, OA DISEASE SEVERITY, AND GAIT MECHANICS**

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**Purpose:** There is increasing evidence that loading at the knee during walking is an important consideration in assessing knee osteoarthritis (OA). While the knee adduction moment (KAM) has been associated with knee OA, it only partially describes the total force at the knee. Recent evidence suggests that analyzing the flexion moment (KFM) in addition to the KAM results in better assessment of the total joint force. Thus, there is a need to better understand the role of the KFM, as the KAM and KFM could influence different regions of the cartilage as well as have different relationships to disease severity.

Thus, the purpose of this study was to test two hypotheses in a medial knee OA population. (1) The magnitude of the peak KAM and KFM are associated with regional cartilage thickness and medial-to-lateral (M/L) cartilage thickness ratios, and (2) the associations between knee moments and cartilage thickness are dependent upon disease severity.

**Methods:** 70 medial knee OA patients (37 female, age: 61.1±8.9 yrs, KL: 2.4±1.2) were stratified by KL grade: grades 1 and 2 were classified as less severe (n=35), and grades 3 and 4 were classified as more severe (n=35). The most painful knee was used for analysis. MR images were acquired on a 1.5T scanner using a sagittal 3D SPGR sequence and segmented to produce 3D cartilage thickness maps. Mean cartilage thickness was computed over the total weight-bearing area of the medial and lateral femoral surfaces and tibial plateaus. The medial and lateral areas were further partitioned into 3 femoral sub-regions (central, internal, and external) and 5 tibial sub-regions (central, internal, external, anterior, and posterior). Subjects also performed a gait test. The first peak KAM and the peak KFM during stance were calculated from each patient and normalized to account for variations in walking speed among individuals. Linear regression analyses tested for associations between regional cartilage thicknesses or M/L thickness ratios and KAM and KFM. The significance level was set at  $\alpha=0.005$  to account for multiple comparisons. To test the second hypothesis, the analyses were repeated separately for the less severe and more severe sub-groups.

**Results:** Only the peak KAM showed a regional dependence with cartilage thickness data in the external, central, and total femoral and tibial regions (Table 1) for all 70 subjects and for the more severe OA patients. In contrast, only the peak KFM was significantly associated with cartilage thickness for less severe OA patients in the posterior and total tibial regions (Table 1). To further assess causes for the role of disease severity on these findings, post-hoc analyses were completed to determine the potential influence of pain. It was found that the magnitude of only the KFM significantly decreased with increasing WOMAC pain score for

'walking on a flat surface' ( $R=-0.272$ ,  $P=0.049$ ) with no significant relationship for the KAM.

**Conclusions:** The results of this study provide new insight into the relationship between regional cartilage degeneration, disease severity, and gait mechanics. For all patients, the KAM had the greatest influence on cartilage thickness, and the primary changes occurred in the medial femoral condyle and M/L thickness ratios in the external and central sub-regions of the femur and tibia. However, the finding of no relationship in the less severe group for the KAM suggests a pathway of pathogenesis for knee OA where a positive association between KAM and cartilage thickness (as previously reported in healthy knees) changes to a negative association that emerges as the disease progresses. The KFM finding is important as it suggests that the KAM does not completely describe the loading environment at the knee. Furthermore, the finding that the KFM was associated with tibial cartilage thickness only in the less severe subgroup suggests the importance of pain in modulating knee loading. Specifically, the post-hoc finding that the peak KFM decreased as pain increased helps to explain why the KFM had no influence on cartilage thinning in the more severe patients. Finally, the regional dependence found in this study suggests that the KAM and KFM may influence different regions of the cartilage at different stages of the disease, thus improving our fundamental understanding of OA development. Together, the results of this study suggest that the severity of the disease and associated pain should be considered when selecting and evaluating interventions for OA that modify knee loading (KAM and KFM).

**Methods:** An asymptomatic subpopulation of subjects with cartilage defects and no self-reported chronic pain or injury to the lower extremity was identified from a larger study (study BMI 18–46 kg/m<sup>2</sup>, ages 20–60 years). As part of the study, bilateral knee magnetic resonance images (MRI), including 3D SPGR and fat-suppressed proton density-weighted sequences, were scored by a radiologist using a method described in the literature. Partial or full cartilage defects in the patellofemoral or tibiofemoral joints were found in 53 (31 female) of the 177 tested subjects (Asymp-OA group; Table 1). A Control group of 31 subjects (16 female) with no cartilage defects in either limb was used from the same larger cohort for comparison (Table 1). Gait mechanics were captured at a self-selected walking speed. The limb was selected at random for gait analysis of the Control group. In the case of bilateral cartilage defects in the Asymp-OA group, the more affected limb was selected. The following measures were analyzed: maximum knee extension angle (degrees) and moment (% body-weight x height) during terminal stance (Fig 1). Hypotheses testing was controlled for potential confounding factors using multiple linear regressions with Group (Asymp-OA versus Control group) as the main predictor and age, BMI, and walking speed as covariates. Standardized regression coefficients were used to compare effect sizes. A Bonferroni-corrected p-value threshold of  $p = 0.03$  was used.

**Results:** The asymptomatic knees with cartilage defects were less extended and had decreased extension moments during terminal stance relative to healthy controls (Figure 1, Table 2). These observations were based on regression statistics that showed a significant negative

#### Significant associations between peak KFM and KAM and regional cartilage thicknesses.

		FEMUR			TIBIA			FEMUR RATIOS			TIBIA RATIOS			
		eM	cM	M	eM	pM	M	eM/eL	cM/cL	M/L	eM/eL	cM/cL	pM/pL	M/L
All Subjects (n=70)	Peak	–	–	–	–	–	–	–	–	–	–	–	–	–
	KFM	–	–	–	–	–	–	–	–	–	–	–	–	–
	Peak KAM	$R=-0.406$ , $P<0.001$	$R=-0.440$ , $P<0.001$	$R=-0.381$ , $P=0.001$	$R=-0.332$ , $P=0.005$	–	–	$R=-0.453$ , $P<0.001$	$R=-0.438$ , $P<0.001$	$R=-0.414$ , $P<0.001$	$R=-0.392$ , $P=0.001$	$R=-0.434$ , $P<0.001$	–	$R=-0.329$ , $P=0.005$
Less Severe Subjects (n=35)	Peak	–	–	–	–	$R=-0.522$ , $P=0.001$	$R=-0.461$ , $P=0.005$	–	–	–	–	–	$R=-0.485$ , $P=0.003$	$R=-0.537$ , $P=0.001$
	KFM	–	–	–	–	–	–	–	–	–	–	–	–	–
	Peak KAM	–	–	–	–	–	–	–	–	–	–	–	–	–
More Severe Subjects (n=35)	Peak	–	–	–	–	–	–	–	–	–	–	–	–	–
	KFM	–	–	–	–	–	–	–	–	–	–	–	–	–
	Peak KAM	$R=-0.579$ , $P<0.001$	$R=-0.548$ , $P=0.001$	$R=-0.499$ , $P=0.002$	–	–	–	$R=-0.557$ , $P=0.001$	$R=-0.511$ , $P=0.002$	$R=-0.480$ , $P=0.004$	–	$R=-0.532$ , $P=0.001$	–	–

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#### ASYMPTOMATIC SUBJECTS WITH MRI-BASED INDICATIONS OF KNEE OA HAVE ALTERED GAIT MECHANICS

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**Purpose:** It is known that cartilage defects associated with knee osteoarthritis (OA) can be present without symptoms. While this early stage of OA is critical in disease etiology, there is little known about early OA because detecting cartilage defects in asymptomatic patients is an unsolved problem. One of our recent studies of healthy subjects yielded a subpopulation of asymptomatic subjects with substantial defects in knee cartilage (Asymp-OA group), therefore providing a unique opportunity to better understand the role of knee mechanics in OA initiation. Previous research has shown gait differences in symptomatic OA patients, specifically decreased knee extension angle and moment peaks, when compared to healthy subjects. However, it remains unclear if these gait differences occur before the disease becomes symptomatic, thus possibly playing a role in OA initiation, or occur at a later disease stage. Therefore, the purpose of this study was to test the hypotheses that, compared to asymptomatic subjects without cartilage defects (Control group), the Asymp-OA knees would H1) be less extended in terminal stance and H2) have a smaller terminal stance extension moment.

predictive effect for "Group" for both the maximum terminal stance extension angle and moment ( $\beta=-0.26$  and  $-0.27$ , respectively; Table 2). Additionally, speed was a significant predictor for maximum terminal stance extension angle ( $\beta=0.24$ ,  $p=0.03$ ).

**Conclusions:** The results indicate that asymptomatic individuals with cartilage defects display altered gait mechanics. Importantly, the altered gait mechanics are consistent with previously-reported gait differences between healthy subjects and patients with knee OA. Further, similar changes in sagittal plane gait mechanics have been associated with knee pain. Taken together with previous literature, the data reported here suggest that there are specific gait characteristics that precede the development of OA symptoms. The cause and effect of these gait alterations warrant future consideration both as a signal for early detection as well as an important opportunity for disease intervention.

Table 1. Study Group Demographics

Measure:	Asymptomatic-OA		Control	
	Mean±SD	Range	Mean±SD	Range
Height (m)	1.7±0.1	1.5–1.9	1.7±0.1	1.6–1.9
Weight (kg)	84.8±19.0	57.6–130.6	74.7±16.1	50.8–108.4
BMI (kg/m <sup>2</sup> )	29±6	19–46	25±4	19–40
Age (years)	43±11	23–60	35±11	20–56